Objectives

- Explain the processes of ventilation and respiration.
- Review conditions caused by pulmonary disease
- Review pleural abnormalities
- Review plural vascular disorders and thoracic surgery

Pathophysiology of Gas Exchange

OXYGENATION AND VENTILATION
Conducting Airways

- Upper airways: warm, humidify, and cleanse air
- Epiglottis: protects opening to the trachea
- Trachea
  - Begins at cricoid cartilage
  - Ends at carina: level of aortic arch, fifth thoracic vertebra, just below the angle of Louis
  - 16 to 20 C-shaped rings to prevent tracheal collapse

Bronchial Tree

- Bronchi:
  - Right – wider and more angular
  - Most common site of foreign body aspiration
  - Left- narrower, positioned above the heart
  - Branches increase area of ventilation despite decreasing airway lumens
- Bronchioles:
  - Gas exchange begins here and continues in alveoli
  - Diameter of less than 1 mm in size
  - Smooth muscle allows for contraction/constriction
  - 32,000 terminal bronchioles

Bronchial Tree

Intrapulmonary section begins after split and forms two main bronchi (Right/Left)
Respiratory Airways
• Respiratory bronchioles: conducting airways and some gas exchange
• Alveoli and epithelial cells

Dead Spaces
• **Alveolar Ventilation** - participates in gas exchange
• **Alveolar Dead Space** – not participating in gas exchange
• **Anatomic Dead Space** – conducting airways (nose, pharynx, bronchi, bronchioles, etc)
  – In disease states, dead space may be higher due to poorly perfused alveoli

Pulmonary Circulation
• Largest vascular bed in body and the ONLY one that received the ENTIRE cardiac output
• Begins at the pulmonary artery (PA) with deoxygenated blood and continues to branch until it forms capillaries that surround alveoli
  • Gas exchange happens
• Oxygenated blood returns to the heart through the pulmonary veins
• Has systolic (PAS) and diastolic (PAD) pressure
  • Can also experience (pulmonary) hypertension
Respiration = Gas Exchange by Diffusion

- From the alveoli into the pulmonary capillary blood to the tissues
- Determined by:
  - Available surface area, open alveoli
  - Integrity of alv-cap membrane
  - Amt. of Hgb in blood
  - Diffusion coefficient of gas and contact time
  - Driving pressures forcing gas to diffuse across membranes
  - Diffusion of gases in and out of cells at the tissue level

Gas Transport to and from Tissue Cells

- Dependent on 4 things:
  - Oxygen content of Hgb – carries 4 molecules
  - Hgb's willingness to give up O2 molecule - Oxyhemoglobin dissociation curve
    - Shifting of curve by temperature, pH, PaCO₂, and 2,3-diphosphoglycerate
    - Shift to the right (better)
    - Shift to the left
  - Abnormalities of hemoglobin – low number or misshapen
  - Carbon dioxide content transported back to lungs in plasma, bound to Hgb or dissolved as carbonic acid

Ventilation

- Ventilation: created by pressure changes
  - Inhalation: active
  - Exhalation: usually passive
- Work of breathing
  - Restrictive lung diseases
  - Obstructive lung diseases
- Regulated by brainstem
Ventilation vs. Perfusion: Gravity

- **Distribution of ventilation**
  - Not even throughout lungs due to gravity and intrapleural pressures
  - On inhalation, the alveoli at the base of the lung receive 4 times more ventilation than the apex

- **Distribution of perfusion**
  - Preferential blood flows to gravity-dependent areas with high gravity pressures in the capillaries in the bases than the apex
  - Intraalveolar pressure is highest in the apexes than the bases and has the potential to exceed capillary hydrostatic pressure resulting in an absence of blood flow to these areas

**Zone I:** potentially little or no perfusion
Ventilation > Perfusion

**Zone II:** varying perfusion
Ventilation = Perfusion

**Zone III:** gravity dependent, well perfused
18 times more blood than apex!
Ventilation < Perfusion

“Good Lung Down”

- Positioning patient with the “diseased lung up” and “good lung down” allows alveoli in diseased lung to open and receive blood flow thereby reducing the V/Q Mismatch
Ventilation/Perfusion: Mismatches Due to Constriction & Shunting

- Distribution of blood is affected by the amount of oxygen in the alveoli.
- Most blood vessels will dilate in response to hypoxia, pulmonary vessels with constrict when PaO\textsubscript{2} is less than 60mmHg = hypoxic vasoconstriction.
- As a result, the pulmonary capillary constricts, “shunting” its blood to another area that has working alveoli.

Definitions

- **Hypoxemia** - reduced oxygenation of arterial blood (PaO\textsubscript{2}) caused by respiratory alterations
  - Low PO\textsubscript{2} (inspired gas)
  - Hypoventilation (low RR)
  - Diffusion abnormality
  - V/Q mismatch
  - Shunting

- **Hypoxia** – reduced oxygenation of cells in tissues, occurs anywhere in body
  - Hypoxemia can lead to hypoxia
  - Hypoxia can result from other abnormalities

- **Hypercapnia** – increased CO\textsubscript{2} in arterial blood (PaCO\textsubscript{2}) caused by alveolar hypoventilation

Acute Lung Injury

- Respiratory Distress and Failure
- Atelectasis
- Pulmonary Edema
- Transfusion Related

**CONDITIONS CAUSED BY PULMONARY DISEASE OR INJURY**
Acute Lung Injury (ALI)

- Non-cardiac pulmonary edema and disruption of the alveolar-capillary membrane resulting from injury to:
  - Pulmonary vasculature
  - Airways or lung tissue
- The most severe form of ALI is Acute Respiratory Distress Syndrome (ARDS)

Risk Factors for Acute Lung Injury

- Direct injury
  - Aspiration
  - Near-drowning
  - Toxic inhalation
  - Pulmonary contusion
  - Pneumonia
    - Interstitial pneumonitis
  - Oxygen toxicity
  - Transthoracic irradiation
- Indirect Injury
  - Sepsis
  - Non thoracic trauma
  - Hyper transfusion
  - CABG
  - Severe pancreatitis
  - Embolism
  - Disseminated intravascular coagulation (DIC)
  - Shock states

Atelectasis

- Collapse of lung tissue
  - malignancy, fluid, air in pleural space or abdominal distention
  - hypo ventilated alveoli or inhalation of concentrated O2 and anesthesia
- Common post-op!!
Postoperative Atelectasis

- Shallow breathing due to pain, fluid and mucous pools dependent lungs

- **Prevention**
  - Frequent turning, postural drainage, deep breathing, early ambulation, air humidification, and incentive spirometry

- **Treatment**
  - Corrected by deep breathing, coughing!!

---

**Pulmonary Edema**

**Excess fluid in lungs**

- Kept dry by lymphatic drainage, oncotic pressures and surfactant (water repellant)

- **Causes**
  - **Overhydration with IV fluids**
  - **Heart failure** — higher filling pressure in LV causes effect in pulmonary system that pushes fluid to interstitial space between alveolus and capillary > overwhelms lymph system
  - **Injury to capillary endothelium** — ARDS or inhaled toxins, hypoalbuminemia
  - **Blocked lymph system** — compressed lymph vessels, edema, malignancy

---

**Pulmonary Edema**

[Diagram showing the causes and effects of pulmonary edema]

1. Vascular dysfunction
   - Coronary artery disease
   - Left-ventricular dysfunction

2. Injury to capillary endothelium

3. Leakage of pulmonary vessels

- Increased left atrial pressure
- Increased capillary pressure

- Movement of fluid and plasma proteins from capillary to interstitial space

- Accumulation of fluid in interstitial space
Pulmonary Edema
Treatment

• Treatment Goals
  – Remove offending agent
  – Supportive therapy to maintain oxygenation and ventilation
    • High Flow oxygen
    • CPAP with Positive end expiratory pressure (PEEP), Mechanical ventilation
  – Move fluid out of lungs - Diuretics
  – Vasodilators – Morphine, Nitroglycerin

Definitions

• Respiratory Distress
  – Increased work of breathing (ventilator effort)

• Respiratory Failure
  – Clinical condition in which there is inadequate blood oxygenation and / or ventilation to meet the metabolic demands of body tissue

Acute Respiratory Failure

Lungs

Respiratory pump

Pulmonary Failure
• $\text{PaO}_2$ ↓
• $\text{PaCO}_2$ ↑

Hypoxic Respiratory Failure

Ventilatory Failure
• $\text{PaO}_2$ ↓
• $\text{PaCO}_2$ ↑

Hypercapnic Respiratory Failure
Respiratory Failure

Get arterial blood gas to diagnose

- $\text{PaO}_2 < 60 \text{ mm Hg} = \text{hypoxia}$
- $\text{PaCO}_2 > 45 \text{ mm Hg} = \text{hypercapnia}$
- In patients with chronically elevated $\text{PaCO}_2$ levels, the pH is also included ($\text{pH} < 7.35$) = hypercapnia with respiratory acidosis

Treatment

- Correct respiratory acidosis!
  - Sodium bicarbonate not recommended even with pH less than 7.2

Acute (Lung) Failure

- Results in:
  - **Alveolar hypoventilation** – not enough $\text{O}_2$ coming in to meet demands = high $\text{CO}_2$
    - Slow RR
  - **Ventilation/perfusion (V/Q) mismatch** – alveoli are collapsed or partially filled with fluid so diffusion across PCM is inadequate for tissue demands
    - atelectasis
  - **Intrapulmonary shunting** – worsening V/Q mismatch. Unoxygenated blood (shunted) mixes with oxygenated blood = lower overall average $\text{O}_2$ going to tissues = begins lactic acid buildup and organ dysfunction
    - Really bad atelectasis or edema!
Acute Respiratory Distress Syndrome

- Characteristics
  - Acute lung inflammation
  - Diffuse alveolocapillary injury
  - Injury to the pulmonary capillary endothelium
  - Inflammation and platelet activation
  - Surfactant inactivation
  - Atelectasis

- Signs and Symptoms
  - Hypoxemia unresponsive to increasing concentration of oxygen!!
  - Rapid, shallow breathing = respiratory alkalosis
  - Continues to progress to respiratory acidosis
  - Hypotension -> death!

Initial Management – Acute Respiratory Distress

- Call for HELP!!
- Airway
  - Clear airway
  - Keep open with head tilt / chin lift, insert Oral/Nasal airway as needed
- Breathing
  - Provide O2 via non-rebreather mask at 15L and / or assist breathing with Ambu bag
  - Monitor O2 sats
  - Prepare for endotracheal intubation
- Circulation
  - Obtain VS and monitor HR, Rhythm, BP every 5-15 min.
  - Provide NS bolus per ESO’s as needed
  - Provide vasoactive medications per MD order

Acute Respiratory Distress Syndrome

- Fulminant form of respiratory failure
  - “White out” on CXR

![Normal](Normal) ![ARDS](ARDS)
Treatment

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Common Clinical Cause</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low PaO₂ (inspired oxygen)</td>
<td>High altitude</td>
<td>Add supplemental oxygen</td>
</tr>
<tr>
<td></td>
<td>Low O₂ content of air suffocation</td>
<td></td>
</tr>
<tr>
<td>Hypoventilation (Low RR, high CO₂)</td>
<td>Lack of neurologic stimulation (over sedation)</td>
<td>Increase rate and depth of breathing</td>
</tr>
<tr>
<td></td>
<td>COPD</td>
<td></td>
</tr>
<tr>
<td>Alveolocapillary diffusion abnormality</td>
<td>Emphysema, Fibrosis</td>
<td>Improve time for diffusion, reduce edema</td>
</tr>
<tr>
<td></td>
<td>Edema</td>
<td></td>
</tr>
<tr>
<td>Ventilation-Perfusion Mismatch</td>
<td>Asthma</td>
<td>Open alveoli, decrease fluid</td>
</tr>
<tr>
<td></td>
<td>Chronic bronchitis, pneumonia</td>
<td></td>
</tr>
<tr>
<td>Shunting</td>
<td>ARDS</td>
<td>Add positive pressure to expose alveoli to capillary bed</td>
</tr>
<tr>
<td></td>
<td>Atelectasis</td>
<td></td>
</tr>
</tbody>
</table>

Facilitate Ventilation – Respiratory Pump

- Erect positioning - Gravity!!
  – Lean forward or side to side for chronic lung disease
  – Prone for acute respiratory failure
- Add pressure to overcome the dead space!!
  – Noninvasive ventilation (CPAP, BiPAP)
  – Invasive mechanical ventilation
Managing Acute Respiratory Failure

- **Oxygenation**
  - Oxygen therapy is given to correct hypoxemia
  - Maintain oxygen saturation at more than 92%

- **Ventilation**
  - Add pressure to overcome the dead space!!
    - Noninvasive ventilation (CPAP, BiPAP)
    - Invasive mechanical ventilation

- **Pharmacology**
  - Bronchodilators: open airways
  - Sedatives: comfort, decrease work of breathing
  - Analgesics: pain control

Nursing Management

- **Positioning**
  - Position patient to best match V/Q
  - Place good lung down
  - Reposition at least every 2 hours

- **Preventing desaturation**
  - Procedures only when needed
  - Hyperoxygenate before suction
  - Provide rest and recovery time
  - Minimize oxygen consumption
    - Limit physical activity
    - Sedation to control anxiety
    - Control fever
  - Continuous pulse oximetry monitoring

Transfusion Related Acute Lung Injury

- **Transfusion Associated Circulatory Overload (TACO)**
- **Transfusion Associated Acute Lung Injury (TRALI)**
Transfusion Associated Circulatory Overload (TACO)

- Caused by administering the transfusion faster than the circulatory system can accommodate

Symptoms include:
- Cough
- Dyspnea
- Pulmonary congestion
- Hypoxia
- Bilateral pulmonary infiltrates
- Tachycardia
- JVD
- Hypertension

TACO Treatment

- Slow transfusion
- Contact MD
- Position patient upright with feet in dependent position
- Administered prescribed diuretics, oxygen and morphine
- Obtain BNP chemistry
Transfusion Services can divide blood unit into smaller amounts to allow for decrease in IV intake

Transfusion Associated Acute Lung Injury (TRALI)

- New acute lung injury (ALI)/acute respiratory distress syndrome (ARDS) occurring during or within six hours after blood product administration
- Caused by reaction to anti-leukocyte antibodies in blood or blood component
- Associated with all blood products, high-plasma-volume components such as plasma, apheresis platelet concentrates, and whole blood have been consistently shown to carry the greatest risk per component or per transfusion episode

Transfusion Associated Acute Lung Injury (TRALI)

- Risk factors
  - Liver transplantation surgery
  - Chronic alcohol abuse
  - Shock
  - Higher peak airway pressure while being mechanically ventilated
  - Current smoking
  - Higher interleukin (IL)-8 levels
  - Positive fluid balance
TRALI Symptoms

- Symptoms
  - Dyspnea
  - Hypoxemia
  - Bilateral pulmonary edema
  - Hypotension
  - Fever, chills
  - Shock

- Treatment
  - Support lung function: oxygen, BiPAP and or mechanical ventilation
  - Maintain hemodynamic parameters; may need IV vaspressors
  - Steroids

TRALI

- Obtain VS, O2 sats and / or ABG
- STOP BLOOD TRANSFUSION
- Keep IV open with new bag of NS attached to IV hub
- Initiate transfusion reaction work-up
  - Draw 6mL of blood place in EDTA tube, label with patients name and MRN, and Post #1
  - Primary MD to contact Transfusion Services Physician for consultation
  - Send blood bag and tubing sealed to Transfusion Services for analysis

Pneumothorax
Pleural Effusion

PLEURAL ABNORMALITIES
Pleural Abnormalities - Pathophysiology

• **Pneumothorax**
  – Air in pleural space compresses lung tissue (alveoli)
  – V/Q mismatch and intrapulmonary shunting result

• **Barotrauma**
  – Air travels from pulmonary interstitium to other thoracic structures

**Assessment and Diagnosis**

• **Pneumothorax**
  – Symptoms increase as more lung collapses
  – Larger pneumothorax = greater respiratory distress
  – Absent breath sounds over affected area
  – ABG: hypoxemia and hypercapnia
  – Chest radiograph: confirm pneumothorax

• **Barotrauma**
  – Symptoms are more subtle
  – Air in subcutaneous tissues
  – Air in mediastinal space causes stabbing pain
  – Chest radiograph: confirm barotrauma
Medical Management

• Pneumothorax less than 15%
  – No interventions are needed
• Pneumothorax greater than 15%
  – Requires evacuation of air from pleural space
    • Heimlich valve
    • Chest tube

Tension Pneumothorax

Site of rupture acts like one-way valve. Air enters on inhalation, doesn’t escape on exhale
• Life threatening!
• Mediastinum, heart and great vessels are displaced
• Emergent needle decompression
• Chest tube insertion

Pneumothorax

• Nursing management
  – Optimize oxygenation and ventilation
    • Oxygen to Treat Pneumothorax Protocol for RT
  – Manage the chest tube system – Refer to UCSD Pulmonary Clinical Practice Guidelines
• Promote evacuation of air by encouraging coughing and deep breathing and using Incentive Spirometer
• Splint chest with pillow to enhance respiratory excursion
• Have patient sit up in High Fowler’s to facilitate lung expansion
Pleural Effusion

• Fluid in the pleural space
• Signs
  – Identified by CXR, CT Scan, Ultrasound
• Thoracentesis
  – Drainage of pleural fluid
  – Re-inflation of the lung
• Malignant pleural effusion
  – Goal for malignant pleural effusions is to improve quality of life and reduce symptoms
  – Pleurex Catheter

Pleural Effusion - Nursing Management

• Supplemental oxygen may ease the patient’s symptoms by improving hypoxemia and reducing the workload of the heart and lungs
• Encourage cough and deep breathing to facilitate drainage
• Pain medication to manage pain and anxiety
• Bundle care to conserve energy
• Sit in High Fowler’s to facilitate lung expansion
PleurX Catheter Drainage System

• Catheter is placed in interventional radiology department
• Used to drain fluid from chest (pleural effusions) or abdomen (ascites) to relieve pressure, pain and facilitate breathing from home
  – Sometimes the patients are admitted for drainage in the hospital
  – Nursing can drain the catheter with the right equipment

Nursing Care

• Obtain order from MD for frequency and amount of drainage.
• Pulmonary Drain
  – Only 1 liter drainage per day to avoid hemodynamic instability with changes in fluid status
  – Perform drainage using Atrium chest drain or PleurX bottle kit as indicated
• Abdominal Drain
  – Only 2 liters per day to avoid hemodynamic instability with changes in fluid status
  – Perform drainage using PleurX kit as indicated